Chronic intake of potato chips in humans increases the production of reactive oxygen radicals by leukocytes and increases plasma C-reactive protein: a pilot study[1–3]

Marek Naruszewicz, Danuta Zapolska-Downar, Anita Kośmider, Grażyna Nowicka, Małgorzata Kozłowska-Wojciechowska, Anna S Vikström, and Margareta Törnqvist

ABSTRACT

Background: Relatively high concentrations of acrylamide in commonly ingested food products, such as French fries, potato chips, or cereals, may constitute a potential risk to human health.

Objective: The objective of this pilot study was to investigate the possible connection between chronic ingestion of acrylamide-containing potato chips and oxidative stress or inflammation.

Design: Fourteen healthy volunteers (mean age: 35 y; 8 women and 6 smokers of >20 cigarettes/d) were given 160 g of potato chips containing 157 mg acrylamide daily for 4 wk.

Results: An increase in acrylamide-hemoglobin adducts in blood was found in all the study subjects, with a mean of 43.1 pmol $\cdot$ L^{-1} $\cdot$ g^{-1} hemoglobin (range: 27–76; $P < 0.01$) in nonsmokers and 59.0 pmol $\cdot$ L^{-1} $\cdot$ g^{-1} hemoglobin (range: 43–132; $P < 0.05$) in smokers. Concurrently, a significant increase ($P < 0.01$) in the oxidized LDL, high-sensitivity interleukin-6, high-sensitivity C-reactive protein, and γ-glutamyltransferase concentrations was observed in both smokers and nonsmokers. A significant increase in reactive oxygen radical production by monocytes, lymphocytes, and granulocytes and an increase in CD14 expression in macrophages ($P < 0.001$) were found after intake of potato chips. Twenty-eight days from the discontinuation of the experiment, the variables under study decreased to some extent. It has been shown also that acrylamide increases the production of reactive oxygen species in isolated human monocyte-macrophages in vitro and decreases the cellular glutathione concentration.

Conclusion: These novel findings seem to indicate that chronic ingestion of acrylamide-containing products induces a proinflammatory state, a risk factor for progression of atherosclerosis.   *Am J Clin Nutr* 2009;89:773–7.

INTRODUCTION

The published data pertaining to the effects of acrylamide on human health have focused on the influence of this compound on the function of the central and peripheral nervous systems (1). Most investigations of acrylamide toxicity have concerned themselves with the pure chemical form of the substance. Occupational exposure to acrylamide in chemical industry employees, as well as acrylamide intake in potable water or in cigarette smoke, have been studied (2). Swedish researchers reported that acrylamide could be formed in various heat-treated, carbohydrate-rich foods (3, 4). A particularly high concentration of acrylamide was found in potato chips, breakfast cereals, and crisp bread (5). It has been shown that acrylamide content in food results from heat-induced reactions between the amino group of the free amino acid asparagine and the carbonyl group of a reducing sugar (6). In humans, acrylamide is absorbed mainly via ingestion or inhalation and forms adducts with hemoglobin, which appear to be useful biomarkers of acrylamide exposure (7). Conjugation with glutathione (GSH) catalyzed by glutathione-*S*-transferase is the main pathway of acrylamide metabolism and detoxification (8).

Environmental factors, such as diet and nutritional habits, have a significant impact on the course of cardiovascular disorders with underlying atherosclerosis. In recent years, nutritional habits have changed significantly and acrylamide-containing products, such as potato chips, have become widely popular (9).

Because the typical consumption of acrylamide-rich foods has increased, it is more likely that the compound will exert its effects within blood vessels. Therefore, studies assessing acrylamide influence on cardiovascular risk factors may be necessary, especially because many in vitro studies indicate that exposure of various cells to acrylamide leads to a significant reduction in their GSH concentration (10). This in turn may contribute to the creation of oxidative stress conditions that aggravate atherosclerosis (11). Therefore, the purpose of our study was to assess the effect of the chronic intake of acrylamide contained in commonly ingested potato chips on the free radical production

in leukocytes and on inflammation assessed through measurements of C-reactive protein (CRP) concentrations.

SUBJECTS AND METHODS

Fourteen healthy volunteers (8 women and 6 men) with a mean age of 35 y (range: 22–56 y) were recruited. In this population, 4 women and 2 men smoked >20 cigarettes/d. Subjects were excluded if they were known to have a history of diabetes, hypertension, or hyperlipidemia or were on any medications, vitamins, or dietary supplements. Pregnant women and those with elevated CRP concentrations >5 mg/L at baseline and a body mass index (BMI; in kg/m^2) >25 were also excluded.

The study period was divided into 3 phases. In the first 2 wk, all study subjects were required to ingest 400 g of boiled potatoes daily with amounts of fat [a mixture of liquid and hardened vegetable fats heated at 180°C for ≈20–30 min; peroxide value (POV) < 1.0 mEq/kg; carbonyl value (COV) = 7.5] and salt corresponding to those contained in commercially produced potato chips. Subsequently, for the next 28 d, the study subjects ingested 160 g of potato chips daily from one manufactured batch purchased from a shop. The potato chips contained 878 kcal, of which 4.3% was energy from protein, 65.6% was energy from fat with a polyunsaturated/saturated fatty acid ratio of 0.51, and 30.1% was energy from carbohydrates, together with 1374 mg of sodium and 5 mg of vitamin E. The acrylamide content was 980 μg/kg (156.8 μg per daily ingested 160-g portion), and the concentration of *trans* fatty acids was <1% (12). The POV value was <1.2 mEq/kg and the COV was 10.1. For the following 28 d, the study subjects were strictly forbidden to eat potato chips, which were replaced once again by boiled potatoes. Throughout the experiment, the study subjects maintained their baseline ingestion level of products that can potentially influence acrylamide concentrations, ie, French fries, coffee, and crisp bread. For the entire duration of the study, the participants remained under medical supervision.

The study was conducted according to the Declaration of Helsinki guidelines and was approved by the Institutional Ethics Board of the National Institute of Food and Nutrition. The study commenced in 2005 and was concluded in 2007.

After routine physical examination, blood samples were drawn after an overnight fast and stored at −70°C. Plasma lipids and lipoproteins, glucose, uric acid, and γ-glutamyltransferase (GGT) were measured by standard techniques. Plasma total homocysteine concentrations were measured by using HPLC with fluorescence detection and test kits from Bio-Rad (Munich, Germany). Serum concentrations of CRP were assessed using a high-sensitivity (hs) method (immunonephelometry; Dade Behring GmbH, Marburg, Germany). Oxidized LDL (ox-LDL) was quantified by using a noncompetitive enzyme-linked immunosorbent assay (Mercodia, Uppsala, Sweden). The CV was <5.5%. Serum concentrations of interleukin-6 (hs IL-6) were measured by quantitative sandwich enzyme-linked immunosorbent assay (R&D Systems, Minneapolis, MN). The limit of sensitivity for the assays was 0.02 mg/dL and the CV was 3.5–4.1%.

The *N*-alkyl Edman method for analysis of adducts to *N*-terminal valines in hemoglobin was used with modifications for analysis of acrylamide adducts (13, 14). Globin was isolated by precipitation from thawed samples of erythrocytes. The globin samples were derivatized with pentafluorophenyl iso-

thiocyanate, leading to detachment of the acrylamide adduct as *N*-(2-carbamoylethyl) valine-PFPTH (AA_Val-PFPTH), which was then extracted from the rest of the protein. The deuterated internal standard, AA-d$_7$Val-PFPTH, was added to the derivative samples before extraction. The analysis was performed by gas chromatography–tandem mass spectrometry (Finnigan TSQ 700; ThermoScientific, Waltham, MA) in the negative ion/chemical ionization mode.

Production of reactive oxygen radicals by monocytes, granulocytes, and lymphocytes was analyzed by burst test (Orpegen Pharma, Heidelberg, Germany) using flow cytometry (FACS Scan; Becton Dickinson, San Jose, CA). Cells were stimulated with the use of phorbol myristate acetate (PMA) and monocytes, granulocytes, and lymphocytes were gated (10^4 cells) on the basis of forward-angle light scatter and single-angle light scatter.

Expression of CD14 antigen on monocytes was determined using fluorescein-5-isothiocyanate–labeled monoclonal antibodies (Becton Dickinson).

Isolation of peripheral blood mononuclear cells and cultivation of monocyte-macrophages, as well as measurement of reactive oxygen species (ROS) production with 2′,7′-dichlorofluorescein, were performed as previously reported (15). Cellular GSH concentrations were determined according to Griffith (16) (**Figure 1**). The results are presented as means ± SDs. Arithmetic means were compared using Student's *t* test and 1- or 2-factor analysis of variance followed by Tukey's test. All calculations were made with the StatSoft statistical package (version 5.1 PC; StatSoft Poland, Warsaw, Poland).

The results obtained for hs CRP were compared using the nonparametric Wilcoxon's test for paired values. Two-sided *P* values <0.05 were considered to be significant.
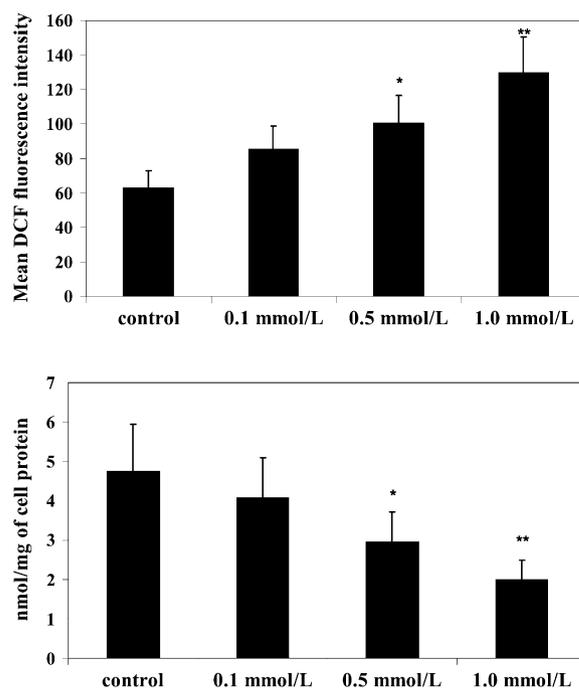

FIGURE 1. The effect of acrylamide on reactive oxygen species production (top panel) and glutathione concentrations (bottom panel) in human-derived monocytes-macrophages in vitro. The results are expressed as means ± SDs from 4 experiments. *, **Significantly different from control (one-factor ANOVA and Tukey's test): *P < 0.01, **P < 0.001. DCF, 2′,7′-dichlorofluorescein.

RESULTS

In all study subjects, significant increases in acrylamide-hemoglobin (AAHb) adducts in blood were found after potato chip intake, which confirms the volunteers' compliance with the experimental conditions. Because the baseline values of AAHb and hs CRP in smokers were significantly higher compared with nonsmokers, the results for the groups are presented separately. As shown in **Table 1**, no changes were detected in BMI, blood pressure (data not shown), or in the plasma concentrations of lipids, lipoproteins, homocysteine, uric acid, or glucose throughout all the experimental periods. There was a significant increase in the concentration of AAHb in nonsmokers ($P < 0.01$) and in smokers ($P < 0.05$) after 28 d of potato chip intake.

In absolute values, the increase in AAHb was significantly larger in smokers than in nonsmokers (mean: 59.0 and 43.1 pmol · L^{-1} · g^{-1} hemoglobin, respectively; $P < 0.05$). A potential interaction of potato chip intake with smoking was also analyzed with 2-factor analysis of variance; however, no significant relations were found. After the 28-d period with no potato chip intake, the concentrations of AAHb were partially reduced yet still remained significantly higher ($P < 0.05$) than the baseline values.

During the potato chip intake period, there was a significant increase in the following concentrations for nonsmokers and smokers, respectively: hs IL-6 concentrations of 20.6% ($P < 0.01$) and 15.1% ($P < 0.01$); ox-LDL concentrations of 21.0% ($P < 0.01$) and 29.1% ($P < 0.01$); hs CRP concentrations of 56.0% ($P < 0.01$) and 43.0% ($P < 0.05$); and GGT concentrations of 21.7% ($P < 0.006$) and 29.6% ($P < 0.01$).

For the evaluation of the oxidative burst activity of leukocytes in heparinized whole blood, flow cytometry and a test kit were used. After the potato chip intake period, a significant increase in reactive oxygen radical production by monocytes, lymphocytes, and granulocytes after stimulation with PMA was observed in both experimental groups (**Table 2**). Furthermore, an increase in CD14 expression in macrophages ($P < 0.001$) was also seen, indicating activation of these cells.

In an independent in vitro study, we demonstrated that 24-h incubation of human monocytes-macrophages in the presence of increasing concentrations of acrylamide (from 0.1 to 1 mmol/L) results in a significant increase in the production of ROS. Simultaneously, we observed a drop in the GSH concentration in those cells.

DISCUSSION

In this study, we have shown for the first time that chronic ingestion of dietary acrylamide might induce oxidative stress in humans through leukocyte activation and increased production of reactive oxygen radicals. As a consequence, a significant increase in the ox-LDL, hs IL-6, and hs CRP concentrations occurred, with all these factors recognized as capable of enhancing atherosclerosis progression. This could be related to the lowering of the reserves of an important cellular antioxidant, GSH, by acrylamide contained in potato chips (17).

Indeed, GSH is used in the process of detoxication and acrylamide removal from the body in the form of mercapturic acid (2). In our study, we actually demonstrated that high acrylamide ingestion is followed by an increase in GGT, which serves as a rescue enzyme for cellular GSH synthesis (18). In the subsequent direct in vitro experiment, we confirmed the actual involvement of acrylamide in the production of ROS by macrophages as well as in the lowering of GSH concentrations in

TABLE 1
Effects of potato chip consumption on acrylamide-hemoglobin (AAHb) adducts, biochemical variables, and BMI[1]

Variables	Nonsmokers ($n = 8$)			Smokers ($n = 6$)		
	Before consumption	28 d after consumption	After 28 d without consumption	Before consumption	28 d after consumption	After 28 d without consumption
AAHb (pmol/g globin)	43.88 ± 31.30	87.00 ± 47.05[2]	74.38 ± 50.61[3]	202.83 ± 185.81[4]	261.83 ± 191.07[3]	230.00 ± 175.71[3]
TC (mmol/L)	5.11 ± 0.92	5.39 ± 1.00	5.19 ± 0.98	5.41 ± 0.76	5.29 ± 0.70	5.10 ± 0.92
TG (mmol/L)	1.12 ± 0.80	1.10 ± 0.51	1.08 ± 0.61	1.49 ± 0.58	1.37 ± 0.49	1.47 ± 0.72
HDL (mmol/L)	1.54 ± 0.28	1.64 ± 0.34	1.42 ± 0.40	1.45 ± 0.36[5]	1.51 ± 0.41	1.51 ± 0.27
LDL (mmol/L)	3.05 ± 1.05	3.29 ± 0.98	3.27 ± 1.09	3.28 ± 1.08	3.16 ± 0.99	3.13 ± 1.11
ox-LDL (U/L)	68.35 ± 15.92	82.7 ± 12.85[2]	73.21 ± 11.52	76.81 ± 12.52[5]	99.18 ± 15.31[2]	80.23 ± 13.28
hs IL-6 (ng/mL)	3.25 ± 0.22	3.92 ± 0.38[2]	3.18 ± 0.41	4.18 ± 0.75[6]	4.81 ± 0.69[2]	4.23 ± 086
hs CRP (mg/L)	1.53 ± 0.75	2.39 ± 0.78[2]	1.79 ± 0.81	2.03 ± 0.73[6]	2.92 ± 0.59[3]	2.25 ± 0.71
GGT (mg/L)	25.56 ± 3.27	33.86 ± 3.95[2]	28.01 ± 2.90	30.53 ± 5.41[5]	38.97 ± 5.19[3]	33.17 ± 3.26
Uric acid (μmol/L)	291.44 ± 67.35	297.40 ± 61.39	295.62 ± 76.29	298.00 ± 76.29	292.04 ± 116.22	286.10 ± 89.40
Homocysteine (μmol/L)	9.86 ± 2.69	10.17 ± 3.45	9.82 ± 3.07	10.96 ± 1.94	10.61 ± 2.02	10.22 ± 2.26
Glucose (mmol/L)	4.93 ± 0.35	5.02 ± 0.24	5.14 ± 0.43	4.92 ± 0.34	5.02 ± 0.28	5.11 ± 0.30
BMI (kg/m^2)	23.06 ± 1.88	23.15 ± 2.01	23.07 ± 1.95	23.8 ± 1.6	23.6 ± 2.1	24.1 ± 1.3

[1] Values are means ± SDs. TC, total cholesterol; TG, triacylglycerol; ox-LDL, oxidized LDL; hs IL-6, high-sensitivity interleukin-6; hs CRP, high-sensitivity C-reactive protein; GGT, γ-glutamyltransferase. Interactions of chips intake with smoking: no significant relations (2-factor ANOVA and Tukey's test).

[2] $P < 0.01$ compared with before consumption (one-factor ANOVA and Tukey's test).

[3] $P < 0.05$.

[4] $P < 0.001$ compared with nonsmokers before consumption (Student's t test).

[5] $P < 0.05$.

[6] $P < 0.01$.

TABLE 2
Effects of potato chip consumption on reactive oxygen radicals in leukocytes after stimulation with phorbol myristate acetate and CD14 expression in monocytes[1]

Variables	Nonsmokers ($n = 8$)			Smokers ($n = 6$)		
	Before consumption	28 d after consumption	After 28 d without consumption	Before consumption	28 d after consumption	After 28 d without consumption
Monocytes	46.38 ± 8.38	99.00 ± 36.93[2]	67.75 ± 20.06[2]	46.67 ± 6.74	87.83 ± 27.36[2]	72.17 ± 19.99[2]
Lymphocytes	17.50 ± 2.62	33.63 ± 11.82[2]	24.13 ± 6.53[2]	19.33 ± 2.34	34.00 ± 9.10[2]	24.83 ± 3.66[2]
Granulocytes	84.13 ± 55.40	141.50 ± 58.2[2]	115.00 ± 55.93[2]	63.33 ± 13.37	115.50 ± 28.02[2]	82.00 ± 8.02[2]
CD14 in monocytes	505.00 ± 52.14	599.13 ± 60.91[3]	582.13 ± 59.96[3]	576.33 ± 131.13	679.17 ± 121.44[4]	591.33 ± 92.00

[1] Values are means ± SDs. Results are shown in units of fluorescense intensity for 10^4 cells. For smokers compared with nonsmokers before consumption, there were no significant differences (Student's t test). For interactions of potato chip intake with smoking, there were no significant relations (2-factor ANOVA and Tukey's test).

[2] $P < 0.05$.

[3] $P < 0.01$.

[4] $P < 0.001$ compared with before consumption (one-factor ANOVA and Tukey's test).

these cells. The cellular GSH decrease induced by acrylamide may also be responsible for mechanisms of molecular regulation of genes through influence on transcription factors, including nuclear factor-κB. This is confirmed by the recent studies by Clement et al (19) and Hasegawa et al (20), which were conducted with the use of genomic and proteomic analysis. The fact that acrylamide may contribute to nuclear factor-κB activation seems to explain the increased CD14 expression in the monocytes that we observed, as well as increased ROS production by leukocytes, which is associated with NADPH-oxidase stimulation. A further consequence is the induction of chronic low-intensity inflammation, shown by an elevated concentration of hs CRP, which is an independent marker of coronary heart disease events (21). Nevertheless, further studies are needed to investigate our observation that, despite a stop in potato chip intake, both the AAHb and reactive oxygen radical production in leukocytes showed a tendency to remain to some extent for the next 28 d. This could be associated with a slower rate of AAHb catabolism via the receptor-dependent endocytosis. Indeed, the latest studies by Schaer et al (22) showed that some of the hemoglobin modifications could reduce its uptake via the CD163 receptor. This in turn might have effects on the maintenance of inflammation, because proper hemoglobin clearance is necessary to counteract its cytotoxicity resulting from oxidative and nitrosative processes (23).

These newly described mechanisms of the action of acrylamide and its metabolite glycidamide may also be responsible for progression of cancer, irrespective of the direct effect of these substances on DNA damage (17). Despite many controversies as to the cancer-promoting action of dietary acrylamide, recent prospective studies indicate that the risk of postmenopausal endometrial and ovarian cancer increases with mean acrylamide ingestion of ≈40 μg/d (24).

Our study, which should be regarded as a pilot study, indicates that high-acrylamide ingestion may constitute a new dietary risk factor for atherosclerosis progression, both in a healthy population and in patients with coronary artery disease. This hypothesis is supported by the studies by Ashfaq et al (25), in which low GSH concentration, associated oxidative stress, and increased hs CRP concentrations were shown to correlate with early atherosclerosis development in healthy people, as assessed by carotid intima-media thickness measurements. Moreover, the concentration of GGT increase after acrylamide ingestion may be a prognostic indicator of the risk of ischemic heart disease and diabetes as well as of hypertension, which was demonstrated in the CARDIA (Coronary Artery Risk Development in Young Adults) Study (26). Additionally, it has recently been demonstrated that elevated GGT may be a risk factor for restenosis in vascular stents (27).

Our study was performed under the conditions of an acute experiment in which the concentration of acrylamide ingestion was 3 times higher than the currently calculated ingestion of ≈50 μg/d in the Western diet (9). Furthermore, the study group was relatively small, which is a limitation to the statistical assessment of the results. Therefore, we recognize that the study results require corroboration by other centers. Also we cannot completely exclude the possibility that the increased uptake of partially oxidized lipids, generated in the process of the production of potato chips, could to some extent influence the results obtained in our experiments (28). However, in view of the fact that the values defining the quality of fat in the potato chips used in this study, ie, POV and COV, were within the lower limits of the acceptable range, we consider the probability of their involvement in the induction of oxidative stress to be small.

In conclusion, long-term ingestion of high-acrylamide doses with food may cause chronic inflammation and contribute to the development of early atherosclerosis as well as increase the risk of coronary artery disease.

REFERENCES

1. LoPachin RM, Ross JF, Lehning EJ. Nerve terminals as the primary site of acrylamide action: a hypothesis. Neurotoxicology 2002;23:43–51.
2. Exon JH. A review of the toxicology of acrylamide. J Toxicol Environ Health B Crit Rev 2006;9:397–412.

3. Tareke E, Rydberg P, Karlsson P, Eriksson S, Törnqvist M. Acrylamide: a cooking carcinogen? Chem Res Toxicol 2000;13:517–22.
4. Tareke E, Rydberg P, Karlsson P, Eriksson S, Törnqvist M. Analysis of acrylamide, a carcinogen formed in heated foodstuffs. J Agric Food Chem 2002;50:4998–5006.
5. Becalski A, Lau BP, Lewis D, Seaman SW. Acrylamide in foods: occurrence, sources, and modeling. J Agric Food Chem 2003;51:802–8.
6. Stadler RH, Blank I, Varga N, et al. Acrylamide from Maillard reaction products. Nature 2002;419:449–50.
7. Bergmark E, Calleman CJ, He F, Costa LG. Determination of hemoglobin adducts in humans occupationally exposed to acrylamide. Toxicol Appl Pharmacol 1993;120:45–54.
8. Odland L, Romert L, Clemedson C, Walum E. Glutathione content, glutathione transferase activity and lipid peroxidation in acrylamide-treated neuroblastoma N1E 115 cells. Toxicol In Vitro 1994;8:263–7.
9. Hagmar L, Wirfält E, Paulsson B, Törnqvist M. Differences in hemoglobin adduct levels of acrylamide in the general population with respect to dietary intake, smoking habits and gender. Mutat Res 2005;580: 157–65.
10. Zödl B, Schmid D, Wassler G, et al. Intestinal transport and metabolism of acrylamide. Toxicology 2007;232:99–108.
11. Schulze PC, Lee RT. Oxidative stress and atherosclerosis. Curr Atheroscler Rep 2005;7:242–8.
12. Mojska H, Gielecińska I, Szponar L, Chajewska K. Acrylamide content in potato chips in Poland. Rocz Panstw Zakl Hig 2006;57:243–9.
13. Törnqvist M, Mowrer J, Jansen S, Ehrenberg L. Monitoring of environmental cancer initiations through hemoglobin adducts by a modified Edman degradation method. Anal Biochem 1986;154:255–66.
14. Wirfält E, Paulsson B, Törnqvist M, Axmon A, Hagmar L. Associations between estimated acrylamide intakes, and hemoglobin AA adducts in a sample from the Malmö Diet and Cancer cohort. Eur J Clin Nutr 2008; 62:314–23.
15. Zapolska-Downar D, Siennicka A, Chełstowski K, et al. Is there an association between angiotensin-converting enzyme gene polymorphism and functional activation of monocytes and macrophage in young patients with essential hypertension? J Hypertens 2006;24:1565–73.
16. Griffith OW. Determination of glutathione and glutathione disulfide using glutathione reductase and 2-vinylpiridine. Anal Biochem 1980; 106:207–12.
17. Jiang L, Cao J, An Y, et al. Genotoxicity of acrylamide in human hepatoma G2 (HepG2) cells. Toxicol In Vitro 2007;21:1486–92.
18. Emdin M, Passino C, Michelassi C, et al. Prognostic value of serum gamma-glutamyl transferase activity after myocardial infarction. Eur Heart J 2001;22:1802–7.
19. Clement FC, Dip R, Naegeli H. Expression profile of human cells in culture exposed to glycidamide, a reactive metabolite of the heat-induced food carcinogen acrylamide. Toxicology 2007;240:111–24.
20. Hasegawa K, Miwa S, Isomura K, Tsutsumiuchi K, Taniguchi H, Miwa J. Acrylamide-responsive genes in the nematode Caenorhabditis elegans. Toxicol Sci 2008;101:215–25.
21. Folsom AR, Pankow JS, Tracy RP, et al. Association of C-reactive protein with markers of prevalent atherosclerotic disease. Am J Cardiol 2001;88:112–7.
22. Schaer DJ, Schaer CA, Buehler PW, et al. CD163 is the macrophage scavenger receptor for native and chemically modified hemoglobins in the absence of haptoglobin. Blood 2006;107:373–80.
23. Schaer DJ, Alayash AI, Buehler PW. Gating the radical hemoglobin to macrophages: the anti-inflammatory role of CD163, a scavenger receptor. Antioxid Redox Signal 2007;9:991–9.
24. Hogervorst JG, Schouten LJ, Konings EJ, Goldbohm RA, van den Brandt PA. A prospective study of dietary acrylamide intake and the risk of endometrial, ovarian, and breast cancer. Cancer Epidemiol Biomarkers Prev 2007;16:2304–13.
25. Ashfaq S, Abramson JL, Jones DP, et al. The relationship between plasma levels of oxidized and reduced thiols and early atherosclerosis in healthy adults. J Am Coll Cardiol 2006;47:1005–11.
26. Lee DH, Jacobs DR Jr, Gross M, et al. Gamma-glutamyltransferase is a predictor of incident diabetes and hypertension: the Coronary Artery Risk Development in Young Adults (CARDIA) Study. Clin Chem 2003; 49:1358–66.
27. Ulus T, Yildirir A, Demirtas S, et al. Serum gamma-glutamyl transferase activity: a new marker for stent restenosis? Atherosclerosis 2007;195: 348–53.
28. Naruszewicz M, Woźny E, Mirkiewicz E, Nowicka G, Szostak WB. The effect of thermally oxidized soya bean oil on metabolism of chylomicrons: increased uptake and degradation of oxidized chylomicrons in cultured mouse macrophages. Atherosclerosis 1987;66:45–53.

Erratum

Viña J, Vento M, Garcia-Sala F, et al. L-Cysteine and glutathione metabolism are impaired in premature infants due to cystathionase deficiency. Am J Clin Nutr 1995;61:1067–9.

The units used throughout the article for sulfur amino acid concentrations are incorrect. Instead of ''mmol/L,'' the units should be ''μmol/L.''

doi: 10.3945/ajcn.2009.27739.

Erratum

Selhub J, Morris MS, Jacques PF, Rosenberg IH. Folate–vitamin B-12 interaction in relation to cognitive impairment, anemia, and biochemical indicators of vitamin B-12 deficiency. Am J Clin Nutr 2009,89(suppl):702S–6S.

On page 704S, the third sentence in the second paragraph of the Results and Discussion section included values in parentheses that were erroneously transposed. The sentence should read as follows:

We also noted with interest that, in the Nurses' Health Study, although multivitamin users in the highest plasma vitamin B-12 quartile category preformed somewhat better on cognitive function tests than did subjects who did not use supplements and were in the lowest quartile category for plasma vitamin B-12 (mean difference in global cognitive score: 0.19; 95% CI: –0.01, 0.40), multivitamin users who were, nevertheless, in the lowest vitamin B-12 quartile category performed worse than the comparison subjects (mean difference in global cognitive score: –0.18; 95% CI: –043, 0.07) (21).

In addition, on page 706S, reference 21 should read as follows: Kang JH, Irizarry MC, Grodstein F. Prospective study of plasma folate, vitamin B-12, and cognitive function and decline. Epidemiology 2006;17:650–7.

doi: 10.3945/ajcn.2009.27740.

Erratum

Naruszewicz M, Zapolska-Downar D, Kośmider A, et al. Chronic intake of potato chips in humans increases the production of reactive oxygen radicals by leukocytes and increases plasma C-reactive protein: a pilot study. Am J Clin Nutr 2009,89:773–7.

In the Design section of the Abstract on page 773, the units listed for acrylamide are incorrect. The sentence should read as follows: **Design:** Fourteen healthy volunteers (mean age: 35 y; 8 women and 6 smokers of >20 cigarettes/d) were given 160 g of potato chips containing 157 μg acrylamide daily for 4 wk.

doi: 10.3945/ajcn.2009.27741.